

Comparison of age of first drink and age of first intoxication as predictors of substance use and mental health problems in adulthood*

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Highlights

- Age of First Intoxication, but not Drink, predict adult use disorder outcomes.
- Covariates reduce or nullify associative size; high quality cohorts are needed.
- Age of First Intoxication associated with individual, family and societal metrics.

Abstract

Background: International public policy on age of first alcoholic drink (AFD) has emphasised the long-term benefits of delaying AFD. This study aimed to compare AFD to age of first intoxication (AFI) as predictors of substance use disorder and mental disorder outcomes in adulthood.

Methods: Data were obtained from a longitudinal birth cohort in Christchurch, New Zealand. Participants were born in 1977. Analysis samples ranged from n=1025 (age 18) to n=962 (age 35). Measures of AFD and AFI were generated using parental- and self-report data collected from age 11. Outcomes at age 18-35 were alcohol quantity consumed, DSM-IV alcohol use disorder (AUD) and AUD symptoms, major depression, anxiety disorder, and nicotine, cannabis, and other illicit drug dependence. Covariate factors measured during childhood included family socioeconomic status, family functioning, parental alcohol-related attitudes/behaviours, and individual factors.

Results: There was a significant unadjusted association between AFD and symptoms of AUD ($p < .001$) and nicotine dependence ($p < .05$) but not other outcomes. AFI was significantly ($p < .05$) associated with all outcomes. After adjustment for covariates, the association between AFD and outcomes was not statistically significant. Conversely, in adjusted models, statistically significant ($p < .05$) associations remained between AFI and all AUD and substance use disorder outcomes but not alcohol consumption or mental disorder outcomes.

Conclusions: AFI was a more robust predictor of adult substance use disorder outcomes than AFD. Public health and policy interventions aimed at prevention of long term harms from alcohol should therefore focus on AFI rather than AFD.

Keywords: Age First Drink; Intoxication; Alcohol; AUD; Cohort Study

1. Introduction

1.1 Age of first drinking or age of first intoxication as a public health target

The age of first alcohol drinking (AFD) has traditionally been regarded as an important metric when considering alcohol-related harm in populations (Grant and Dawson, 1997; Schuckit and Russell, 1983). Delaying AFD has been put forward as a public health strategy both to help reduce the harms from alcohol exposure in adolescence and to lower the incidence of later alcohol use disorder (AUD) (Donaldson, 2009; Health and Council, 2009; U.S. Department of Health and Human Services Office of the Surgeon General, 2007). However, it has recently been argued that AFD is not a useful marker of future alcohol problems, and efforts to postpone AFD are unlikely to be effective in preventing later harmful outcomes (Kuntsche et al., 2016). Limitations of AFD as a construct include lack of reliability when ascertained retrospectively in adults, inconsistent definitions, and variable findings on the relationship between AFD and later alcohol and related psychosocial outcomes (for useful overviews, see Hingson et al., 2016; Rossow and Kuntsche, 2013; Maimaris and McCambridge, 2014).

Most short term harms from alcohol use in adolescence result from intoxication rather than alcohol exposure per se. These harms have been extensively studied and are well understood (Feldstein Ewing et al., 2014; Kelly et al., 2015). Rapid progress from first initiation of alcohol use to episodes of intoxication (Morean et al., 2012) and age of first intoxication (AFI) (Monshouwer et al., 2003) have therefore recently attracted interest as markers of the harm from hazardous drinking in adolescents.

In relation to longer term outcomes associated with AFI, studies in college students (Hingson et al., 2003) and in a Native American population (Ehlers et al., 2006) both showed earlier AFI was associated with a higher prevalence of alcohol dependence. However, these studies relied on retrospective ascertainment of AFI. A prospective study, also in a Native

American sample, showed earlier AFI was associated with higher prevalence of alcohol disorder at age 18 (Henry et al., 2011). However, the association between AFI and long term substance use disorder and mental health outcomes in adulthood has still not been well studied in prospective population cohorts.

1.2 Longitudinal approaches to studying the association between AFD/AFI and outcomes

Accurately modelling the putative causal role of AFD or AFI on later outcomes requires longitudinal data and contemporaneous measurement of confounding and covariate factors. These factors include genetic (Young-Wolff et al., 2011), familial (Verhulst et al., 2015), attachment (Haller, 2016), and personality variables (Newton-Howes and Boden, 2016b).

To date, only one prospective study has examined both AFD and AFI in a single cohort (Morean et al., 2012) and found that both AFD and rapid progress from first exposure to first intoxication were risk factors for later alcohol problems. However, this study had several limitations. Its sample was comprised of only college students, there was retrospective questioning about AFD and AFI, and a measure of alcohol problems was the main outcome (rather than alcohol use disorder).

The Christchurch Health and Development Study (CHDS) provides an opportunity to investigate the association between AFD / AFI and later outcomes including alcohol use disorder and mental disorders while controlling for diverse potential confounding variables. The association between AFD and later outcomes has been investigated previously in this cohort (Fergusson et al., 1994b; Newton-Howes and Boden, 2016a). Early examination of AFD in the CHDS cohort showed that earlier AFD was associated with AUD at age 15 (Fergusson et al., 1994b). However, later work on this cohort showed the instability of an adolescent AUD diagnosis (Wells et al., 2006): one half of adolescents with AUD at age 16-

18 did not retain this diagnosis to age 25. More recent work extended previous CHDS analyses by using latent classes of AFD from 0 to 13+ years and measuring outcomes to 35 years (Newton-Howes and Boden, 2016a). This showed no association between child and adolescent AFD and adult addiction and mental health outcomes.

The present study extends these prior analyses on this cohort in two ways: first, it directly compares the predictive effects of AFI against those of AFD; second, it provides a more fine-grained analysis of the exposure variables in adolescence, the age period where AFI usually occurs (Snyder and Monroe, 2013; Van Ryzin et al., 2012).

1.3 Aims of the present study

This study aimed to examine whether AFD or AFI is a more robust predictor of alcohol or other substance disorder and mental disorder outcomes to age 35.

2. Material and methods

2.1 Participants

Data were gathered from the Christchurch Health and Development Study (CHDS), a birth cohort of 1265 individuals born in the Christchurch (New Zealand) urban region in mid-1977 (Fergusson and Horwood, 2001; Fergusson et al., 1989). Sample sizes ranged from 1025 (age 18) to 962 (age 35), which represented 79% to 82% of the surviving sample at each observation.

2.2 Age of first drink

At ages 11, 12 and 13 years, cohort members were asked a series of questions about their experiences of drinking alcohol. One of these questions asked cohort members to report the age at which they recalled first drinking alcohol. From the age of 11 years, cohort members were also asked if they had drunk alcohol in the last year (with a yes/no response option). Further, at each assessment at ages 14, 15, 16, and 18 years, cohort members were asked about their usual frequency of alcohol consumption in the previous 12 months using a

six-point scale ranging from “never” to “almost every day”. Cohort members were also asked about the usual quantity of alcohol consumed (in standard drinks) per drinking occasion over that period by indicating the number and portion size of various drinks including beer, wine and spirits.

In addition to these data, from the age 11 assessment the parents of cohort members were also asked a series of questions about their child’s experiences with alcohol, including the age at which the child consumed alcohol for the first time and whether the child had consumed alcohol during the previous 12 months (to the parent’s knowledge).

Using this information, cohort members were classified on their age of first drink (AFD) based on the age at which alcohol consumption was first reported. Because the specific aims of the study were intended to focus on adolescent use (see introduction), a four-level classification of “≤ age 13”, “age 13-16”, “age 16-18”, and “age 18+” was employed. In cases where there was an affirmative or positive report of alcohol use from either the parent or the child (but not both), the response was deemed to be affirmative.

2.3 Age of first intoxication

From ages 11 to 14, cohort members were asked whether they had ever consumed enough alcohol to become “dizzy or tipsy” during the previous 12 months. Also, at ages 12 to 14, cohort members’ parents were asked whether their child had ever consumed enough alcohol to become “dizzy or tipsy” in the previous 12 months.

From age 15, cohort members were asked a series of questions derived from the Rutgers Alcohol Problem Index. This checklist comprised a series of 30 items describing possible consequences of alcohol consumption, including intoxication.

In addition to these data, from the age 11 (to age 16) assessment, the parents of cohort members were also asked a series of questions about their child’s experiences with alcohol,

including whether the child had ever appeared to be “tipsy or drunk” during the past 12 months (to the parent’s knowledge).

Using this information, cohort members were classified on their age of first intoxication (AFI), based on the age at which intoxication was first reported using a four-level classification of “ \leq age 13”, “age 13-16”, “age 16-18”, and “age 18+”, in order to match that of AFD (above).

2.4 Outcome measures (18-35 years)

2.4.1 Alcohol consumption, problems with alcohol and alcohol use disorders. At each interview from age 18 years onwards, cohort members were asked questions concerning their consumption of alcohol over the past 12 months. Measures included consumption frequency, quantity typically consumed in a single drinking session, and whether the individual experienced any problems relating to their drinking. Alcohol amounts were converted to New Zealand Standard Drinks (10 grams of pure alcohol) and AUD. The latter measure was based on the Composite International Diagnostic Interview (CIDI) (Robins et al., 1995) at ages 18, 21, 25, 30 and 35 years, in order to obtain information pertaining to DSM-IV symptoms of alcohol abuse/alcohol dependence (American Psychiatric Association, 1994). These data provide a) a count measure of the number of symptoms of alcohol use disorder and b) classification of participants as to whether they meet DSM criteria for an alcohol use disorder during each 12 month period following the previous assessment.

2.4.2 Mental health disorders. Cohort members completed the CIDI at ages 18, 21, 25, 30, and 35 years. These data were used to classify individuals as to whether they met DSM-IV criteria for major depression and anxiety disorder over the intervals 18-21 years, 21-25 years, 25-30 years, and 30-35 years. Anxiety disorders included generalized anxiety disorder, panic disorder, agoraphobia, social phobia, and specific phobia.

2.4.3 Other substance use disorders. Also at ages 18, 21, 25, 30, and 35 years, cohort members were questioned about their substance use behaviours and problems associated with substance use since the previous assessment (tobacco, cannabis) based on the CIDI (items for cigarette smoking were custom written; see Supplementary Material¹ for items). Using this information, cohort members were classified as meeting DSM-IV criteria for nicotine dependence and cannabis dependence over the intervals 18-21 years, 21-25 years, 25-30 years, and 30-35 years.

2.5 Potential confounding factors

Several potential confounding factors were included in the analyses on the basis that they were a) used in a prior analysis of AFD in the CHDS cohort (Fergusson et al., 1994b; Newton-Howes and Boden, 2016a) or b) have been found to be related to substance use outcomes in other studies of the CHDS cohort (Fergusson et al., 2008; Fergusson et al., 1995; Fergusson et al., 2007; Fergusson et al., 1993, 1994a; Lynskey et al., 1998). These measures, detailed below, are fully described in the Online Supplementary Material¹.

2.5.1 Measures of family socio-economic and demographic background. Information on maternal age, family living standards (0-10 years), maternal and paternal education, family socioeconomic status (at birth), averaged family income (0-10 years), and New Zealand Maori ethnicity was collected.

2.5.2 Individual, personality and behavioural factors. Factors measured included gender, child conduct problems (7–9 years), neuroticism (age 14), novelty-seeking (age 16), and early onset of cigarette smoking (by age 13).

2.5.3 Family functioning, parental behaviour and abuse exposure measures. Potential confounding factors included were parental illicit drug use (0–11 years), parental alcohol

¹Supplementary material can be found by accessing the online version of this paper at <http://dx.doi.org> and by entering doi:...

problems (0–15 years), parental criminality (0–15 years), parental alcohol consumption (11 years), parental approval of adolescent drinking (15 years), parental attitudes to alcohol use (15 years), changes of parents (to age 15 years), parental attachment (15 years), exposure to harsh/abusive physical punishment (childhood physical abuse; 0–16 years), childhood sexual abuse (0–16 years), parental intimate partner violence (0–16 years).

2.6 Statistical analyses

The associations between AFD/AFI and the repeated measures of alcohol outcomes, mental health and substance use outcomes at ages 18–35 years were modelled separately by fitting population-averaged generalised estimating equation (GEE) models to the data for each outcome (Liang and Zeger, 1986; Zeger and Liang, 1986). For dichotomous outcomes, logistic regression models were fitted; for continuous outcomes, ordinary least squares models were fitted. Adjusted models were fitted by simultaneously entering measures of potential confounding factors noted above. Full details of model fitting are available in the Supplementary Material².

3. Results

3.1 Associations between AFD/AFI and outcomes, ages 18–35

Table 1 shows the cohort divided into groups (≤ 13 ; 13–16; 16–18; 18+) according to their reported AFD/AFI. For each classification, the Table reports on the mean or the percentage for each of the seven outcomes (number of standard drinks consumed, symptoms of alcohol use disorder, alcohol use disorder, major depression, anxiety disorder, nicotine dependence, and cannabis dependence) pooled over the assessment periods 18–21, 21–25, 25–30, and 30–35 years. The Table also reports on the likelihood ratio chi-square test of linear trend for each outcome, derived from bivariate GEE models, for both AFD and AFI. Full data

²Supplementary material can be found by accessing the online version of this paper at <http://dx.doi.org> and by entering doi:...

from each assessment period are shown in the Supplementary Material (Tables S1a to S1g)². In addition, the Spearman Rank Order correlation between AFD and AFI was 0.15 ($p < .0001$). The Table shows that there were statistically significant ($p < .05$) bivariate associations observed between AFD and both symptoms of AUD and nicotine dependence. Those whose introduction to alcohol was delayed tended to have lower rates of AUD symptoms and were less likely to meet criteria for nicotine dependence. For the remaining outcomes, although higher AFD appeared to be related to lower levels of adverse outcomes, none of these associations reached statistical significance.

In contrast, there were consistent statistically significant ($p < .05$) bivariate associations between AFI and each of the seven outcomes. Inspection of the Table shows that, in all cases, higher AFI was associated with decreased risk of adverse outcomes during the period ages 18-35. In all cases, the “18+” AFI group had the lowest rate of each outcome. Further examination of the Tables showed that while most participants were classified according to AFD in the “ ≤ 13 ” group, the majority of participants classified on AFI were in the “18+” group.

It should be noted that, because of the wide variation in reports of amounts of alcohol consumed, the standard deviations for these measures were large.

3.2 Associations between AFD/AFI and potentially confounding factors

One issue arising in the assessment of the associations between AFD/AFI and outcomes is that these associations may be influenced by a series of potentially confounding factors occurring prior to or contemporaneously with AFD or AFI. In order to examine this, a series of measures were drawn from the Study database (see Methods), and Spearman Rank Order Correlations were calculated for each measure and AFD/AFI. These correlations are presented in Table 2 along with tests of statistical significance.

The Table shows that while AFD was correlated with only a subset of potential confounding factors, AFI was significantly ($p < .05$) correlated with all factors.

3.3 Adjustment of associations between AFD/AFI and outcomes

It could be argued that any apparent associations between AFD/AFI and outcomes could be explained by the influence of factors confounded with either AFD or AFI, presented in Table 2 (above). In order to address this issue, the GEE models of the associations between the statistically significant outcomes shown in Table 1 and AFD/AFI were extended to include the potentially confounding individual, demographic, and family factors shown in Table 2. Table 3 shows the parameter estimates for the unadjusted and adjusted associations between AFD/AFI and each of the outcomes and indicates tests of statistical significance. The Table also displays estimates of ΔR^2 (for the measure of amount of alcohol consumed), incidence rate ratio (IRR; for the measure of AUD symptoms), and odds ratio (OR; for dichotomous outcome measures). Estimates of the IRR and OR were computed using the age classification “18+” as the reference category.

The Table shows that after adjustment for confounding factors, AFD was no longer statistically significantly associated with either AUD symptoms or nicotine dependence ($p > .05$). The pattern of results suggested that the bivariate associations between AFD and these outcomes could be fully accounted for by the effects of individual, demographic and family factors associated with a lower AFD.

Adjustment for confounding factors reduced the magnitude of the associations between AFI and all outcomes. However, unlike AFD, after adjustment for confounding factors, five out of the eight outcomes remained statistically significantly ($p < .05$) associated with AFI. These outcomes included AUD symptoms, AUD, nicotine dependence, and cannabis dependence. Amounts of alcohol consumed, major depression, and anxiety disorder were no longer significantly ($p > .05$) associated with AFI after adjustment for confounding.

Estimates of the ΔR^2 , IRR and OR suggest that a) the associations between AFI and outcomes were stronger than those between AFD and outcomes and b) after adjustment, there remained a small to moderate statistically significant association between AFD and several outcomes (AUD symptoms; AUD; nicotine dependence; cannabis dependence).

In general, this pattern of results suggests that AFI is a stronger predictor of substance use problems in adulthood than AFD, although neither measure predicted internalizing disorders (major depression and anxiety disorder) after adjustment for confounding.

4. Discussion

4.1 Main findings

The vast majority of cohort participants were first exposed to alcohol before the age of 13, but over half did not report becoming intoxicated until age 18 or later. Among those whose first exposure to alcohol was in adolescence (age 13-18), earlier AFD was associated with higher unadjusted rates of AUD and nicotine dependence to age 35, but AFD did not predict any other outcomes. No statistically significant associations between AFD and any outcomes were observed in adjusted models.

In unadjusted models AFI showed consistent, moderately strong associations with substance use disorder outcomes. For example, the risk of alcohol dependence at age 18-35 among those with AFI age 13 or younger was approximately twice that of participants with AFI after age 18. In contrast, the unadjusted associations between AFI and major depression or anxiety disorder outcomes were modest. After adjustment for covariate factors, the magnitude of the associations between AFI and substance use outcomes reduced, but it remained statistically significant with the exception of the alcohol consumption outcome. There was no association between AFI and major depression or anxiety disorders in adjusted models.

4.2 Comparison of findings to other literature

The findings extend our earlier analyses on this cohort (Newton-Howes and Boden, 2016a) by adding AFI as an exposure variable and using a more fine-grained measure of AFD in adolescence. The present study suggests AFI is a stronger predictor of later alcohol and other substance use problems than AFD. This may in part relate to cultural practices in this cohort, whereby alcohol exposure before age 13 was normative.

Findings from studies in which AFD was ascertained retrospectively, including in cross-sectional survey data (DeWit et al., 2000; Grant and Dawson, 1997) and longitudinal studies in adolescents (Behrendt et al., 2009; Warner and White, 2003), have suggested earlier AFD is associated with higher levels of later substance use disorder or mental disorder morbidity. However, the strength of evidence in this area has recently been questioned because of marked variation in the way AFD is defined across studies and the poor reliability of retrospective measurement of AFD (Kuntsche et al., 2016). In light of the practical difficulty in delaying AFD in many cultural settings, it has recently been suggested that the length of time between AFD and AFI may be a useful indicator of later alcohol problems and a target for prevention activities (Morean, 2012).

In a related study involving another New Zealand cohort, the Dunedin Multidisciplinary Health and Development Study (Odgers et al., 2008), early substance use predicted substance dependence at age 32. However, the early exposure group in the Dunedin Study comprised about 10% of the cohort who were using a variety of substances by age 15, including alcohol and illicit drugs. Therefore, while the findings are consistent with the CHDS findings, they are not directly comparable to the investigations presented here.

The Australian Parental Supply of Alcohol Longitudinal Study (APSALS) (Aiken et al., 2015) may in the future provide more nuanced answers to the questions posed by the present study, particularly regarding parental supply and specific drinking behaviors (Wadolowski et al., 2015a; Wadolowski et al., 2015b).

4.3 Limitations

AFD and AFI were composite measures developed specifically to examine this research question, and it was not possible to examine in fine detail the progression from first sip of alcohol to more substantive quantities to drinking to intoxication. Furthermore, some of the relatively small numbers of participants in AFD and AFI classes may have led to some imprecision in measurement. The study investigated a cohort born in a particular year in a specific social context. This might limit the generalizability of the findings, although the place of alcohol in New Zealand society is similar to that of many other high-income countries with “wet” drinking cultures. Finally, the reliance on self-report data about alcohol use is a further limitation, although this was likely mitigated by the close relationship built up between investigators and participants during decades of follow up. The main strengths of the present study compared to previous studies in this area are the prospective ascertainment of alcohol exposure from age 11 and the focus on long term outcomes. The birth cohort design also provides the ability to contemporaneously measure a broad range of other factors likely to be confounded with AFD.

4.4 Concluding comments

From a research perspective, future longitudinal studies on the long-term impact of early alcohol use should gather detailed information about the quantity, frequency, and social context of early alcohol exposure from childhood onwards. Measurement should capture episodes of intoxication, because in cultural contexts where early alcohol exposure is normative, the age of first intoxication is likely to predict adult outcomes better than age of first drinking.

From a public health and policy standpoint, delaying the first experience of alcohol intoxication is likely to be a more important and achievable goal than preventing any early exposure to alcohol.

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Conflict of Interest

None declared.

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Table 1. Associations between AFD/AFI and outcomes in the past year, ages 18–35.

Outcome	Age of first drink				Age of first intoxication			
	≤13	13– 16	16– 18	18+	≤13	13–16	16–18	18+
Mean (SD) number of standard drinks	363 (1255)	358 (1145)	144 (206)	310 (1650)	527 (2256)	390 (1072)	476 (1261)	278 (922)
Test of linear trend	$\chi^2(1) = 0.7, p > .40$				$\chi^2(1) = 12.6, p < .001$			
Mean (SD) symptoms of alcohol use disorder	1.73 (4.67)	1.79 (3.30)	1.13 (2.85)	0.75 (2.62)	2.55 (5.91)	2.48 (5.58)	2.45 (5.09)	0.91 (3.05)
Test of linear trend	$\chi^2(1) = 13.7, p < .001$				$\chi^2(1) = 283.3, p < .0001$			
% alcohol use disorder	20.6	18.5	14.8	9.1	28.7	30.3	33.1	14.0
Test of linear trend	$\chi^2(1) = 3.3, p < .10$				$\chi^2(1) = 57.7, p < .0001$			
% major depression	21.3	18.5	26.2	22.7	22.8	27.3	23.5	17.2
Test of linear trend	$\chi^2(1) = 0.1, p > .80$				$\chi^2(1) = 18.4, p < .0001$			
% anxiety disorder	17.1	15.3	9.8	13.6	20.7	18.8	20.9	15.7
Test of linear trend	$\chi^2(1) = 1.5, p > .20$				$\chi^2(1) = 5.2, p < .05$			
% nicotine dependence	22.2	18.0	11.5	2.3	34.8	30.3	20.9	14.1
Test of linear trend	$\chi^2(1) = 5.7, p < .05$				$\chi^2(1) = 60.0, p < .0001$			
% cannabis dependence	5.3	5.8	0.0	2.3	9.1	7.9	6.8	2.3
Test of linear trend	$\chi^2(1) = 1.2, p > .20$				$\chi^2(1) = 40.1, p < .0001$			
n	961	54	17	11	134	272	91	549

Table 2. Spearman's correlations for the associations between AFD/AFI and individual, demographic and family factors measured to adolescence.

	AFD	AFI
Confounding factors		
<u>Family socioeconomic and demographic background</u>		
Maternal age	.10**	.14***
Family living standards ages 0-10	.06	.11**
Maternal education level	.02	.12***
Family SES level at birth	.01	.09**
Average family income rank ages 0-10	.04	.09**
Māori ethnicity at birth	.01	.06*
<u>Individual, personality and behavioural factors</u>		
Female gender	.00	.08*
Conduct problems ages 7-9	-.08*	-.22***
Neuroticism age 14	.03	-.11***
Novelty seeking age 16	-.07*	-.29***
Cigarette smoking by age 13	-.15***	-.28***
<u>Family functioning, parental behaviour and abuse exposure measures</u>		
Parental illicit drug use	-.05	-.14***
Parental alcohol problems	-.01	-.13***
Parental criminality	-.01	-.15***
Parental alcohol consumption age 11	-.19***	-.11**
Parental approval of adolescent drinking age 15	-.20***	-.15***
Changes of parents to age 15	-.03	-.24***
Parental attachment age 15 (Mean)	-.00	.21***

Childhood exposure to harsh/abusive physical punishment	-.03	-.13***
Childhood exposure to sexual abuse	-.05	-.13***
Parental intimate partner violence to age 16	-.07*	-.15***

*p < .05

** p < .01

*** p < .001

Table 3. Parameter estimates for the unadjusted and adjusted associations between AFD/AFI and outcomes (ages 18 – 35).

Outcome	AFD				AFI			
	Model 1		Model 2		Model 1		Model 2	
	Unadjusted		Adjusted for individual, demographic and family factors		Unadjusted		Adjusted for individual, demographic and family factors	
	β /SE	ΔR^2 /IRR/OR	β /SE	ΔR^2 /IRR/OR	β /SE	ΔR^2 /IRR/OR	β /SE	ΔR^2 /IRR/OR
Amount alcohol consumed	- 45.72 (54.58)	.0002	- 10.60 (63.71)	.0001	- 75.16* ** (21.21)	.0043	- 29.28 (26.70)	.0005
Alcohol use disorder symptoms	-.27 (.07)	1.32 (1.07-1.63)	-.03 (.08)	1.03 (0.90-1.20)	- .40*** (.02)	1.49 (1.42-1.57)	- .23*** * (.03)	1.26 (1.19-1.32)
Alcohol use disorder	-.25 (.14)	1.30 (0.98-1.70)	-.09 (.15)	1.09 (0.81-1.48)	- .36*** (.05)	1.43 (1.30-1.55)	- .21*** * (.05)	1.23 (1.12-1.36)
Major depression	.02 (.11)	0.98 (0.79-1.22)	.17 (.12)	0.84 (0.66-1.06)	- .19*** (.04)	1.21 (1.11-1.32)	-.08 (.05)	1.08 (0.98-1.20)
Anxiety disorder	-.18 (.14)	1.20 (0.89-1.58)	-.15 (.15)	1.16 (0.86-1.57)	-.11* (.05)	1.12 (1.01-1.22)	-.01 (.05)	1.01 (0.91-1.13)
Nicotine dependence	-.46* (.19)	1.58 (1.08-2.32)	-.08 (.20)	1.08 (0.73-1.60)	- .43*** (.06)	1.54 (1.38-1.72)	- .19*** (.06)	1.21 (1.06-1.36)
Cannabis dependence	-.29 (.27)	1.34 (0.79-2.25)	.02 (.32)	0.98 (0.52-1.82)	- .51*** (.68)	1.67 (1.42-1.99)	- .29*** (.10)	1.34 (1.09-1.63)

* $p < .05$

** $p < .01$

*** $p < .0001$